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Perihepatitis in women with salpingitis—an under-diagnosed clinical entity?

Perihepatitis, also referred to as Fitz-Hugh-Curtis syndrome (FHCS) is reported to occur in 5 to 15% of women with salpingitis, based on laparoscopic findings.[1] Although *Neisseria gonorrhoeae* was described as the causative organism in the cases reported earlier, *Chlamydia trachomatis* is now recognised as responsible for most cases of perihepatitis.[1] Three patients with FHCS were recognised and treated during a twelve month period in a department of genitourinary medicine.

The first patient was a 27 year old waitress, who was admitted as an emergency to the surgical ward as a suspected case of cholecystitis. She complained of having developed a colicky lower abdominal pain 14 days prior to admission, which, after ten days, migrated to the right upper quadrant. Pain was pleuritic in nature and radiated to the back. She was pyrexial, tender in the right iliac fossa and right hypochondrium and required parenteral analgesics for the relief of pain. Perihepatitis was suspected and she was referred to the genitourinary medicine clinic for confirmation. *C trachomatis* from the cervix was detected. Serological tests for anti-chlamydial antibodies showed the presence of IgM and the microimmunofluorescence (micro-IF) test showed the IgG titre to be > 4096. Perihepatitis was subsequently confirmed at laparoscopy and she responded to a two week course of doxycycline combined with a week's course of metronidazole.

The other two patients presented with similar symptoms but with lesser severity. *C trachomatis* was detected in both instances and their serological tests showed anti-chlamydia IgG titres to be >1024. Both patients responded to therapy with doxycycline. Partner notification was successfully completed in all three instances.

Patients with FHCS can present to a variety of disciplines,[2 3] and the incidence is probably an under estimate. Diagnosis should be suspected in women who are young and who present with right upper quadrant localisation of pain which is pleuritic in nature, associated with uterine and adnexal tenderness on pelvic examination. Detection of *C trachomatis* from the lower genital tract together with the demonstration of high titres of anti-chlamydial IgG (>1:1024) and the presence of IgM antibodies should strongly suggest FHCS.[1]

Although laparoscopic detection of violin string adhesions between the liver capsule and the anterior abdominal wall is essential for confirmation, a recent report has suggested that ultrasound can be used to confirm the diagnosis.[4] If proven, the latter may eventually replace invasive laparoscopic procedures for confirmation of the diagnosis. The role of the genitourinary physician is vital not only to exclude other sexually transmitted diseases and ensure appropriate therapy, but also to treat contacts so that the risk of reinfection is eliminated.

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Importation into the UK of a strain of *Neisseria gonorrhoeae* resistant to penicillin, ciprofloxacin and tetracycline

We report here what we believe to be the first isolation of a strain of penicillinase-producing *Neisseria gonorrhoeae* with high-level resistance to ciprofloxacin and tetracycline.

On the 3 February 1995 a 35 year old divorced male travelled to Angeles City in the Philippines on business. Here he had sexual contact with a local prostitute. He subsequently travelled to Australia by which time he had developed a bloody urethral discharge. He was prescribed 500 mg of tetracycline with 500 000 units of nystatin ("Mysteclin") orally twice daily for 14 days. On return to the Philippines his urethral discharge was still present and he was prescribed 300 mg rosoxacin (a 4-quinolone) *stat* orally. The patient returned to the UK at the end of February with the urethral discharge still present. The patient had had no UK sexual contacts since January. He was examined at his local genitourinary medicine clinic where intracellular Gram-negative diplococci were seen in a smear of the discharge. Urethral swabs were taken for culture and chlamydia antigen assay. He was prescribed 500 mg of ciprofloxacin *stat* and a 10 day course of ofloxacin (400 mg daily) was started.

The urethral swab taken at this time yielded oxidase-positive Gram-negative diplococci after 48 h incubation on New York City medium at 37°C in 5% CO_2. This organism was identified as *N gonorrhoeae* by the carbohydrate utilisation and Phadebact Monoclonal GC tests. The strain was demonstrated to be β-lactamase positive and found by agar dilution antibiotic sensitivity testing to be resistant to penicillin (minimum inhibitory concentration (MIC) >10 mg/l), ciprofloxacin (MIC 16 mg/l) and tetracycline (MIC 64 mg/l) but sensitive to spectinomycin (MIC 32

mg/l) and cefuroxime (MIC 0·32 mg/l). The strain carried plasmids of 3·0MDa and 25·2MDa and the latter was shown to carry the Dutch type *tet*M tetracycline resistance determinant by polymerase chain reaction. Typing studies revealed that the strain belonged to the prototrophic auxotype and the IB7 serovar. The chlamydia antigen assay was negative.

At the end of the course of ofloxacin the patient re-attended the clinic with a persisting urethral discharge. A Gram-stained smear of the discharge revealed intracellular Gram-negative diplococci. Culture of a urethral swab yielded an organism indistinguishable in all respects from the previous isolate. On this occasion the patient was treated with a 2g *stat* IM dose of spectinomycin. On follow up seven days later only a slight discharge was present and both smear and culture were negative for gonococci although polymorphonuclear lymphocytes were seen in the former. He was therefore treated as a case of post-gonococcal urethritis and given a 10 day course of 250 mg oxytetracycline four times daily.

When first introduced ciprofloxacin had exceptional in vitro activity against strains of *N gonorrhoeae*[1-3] and consequently has been increasingly used as a first line treatment for gonorrhoea. However, a treatment failure with another quinolone, enoxacin, was reported some time ago by Wagenvoort *et al.*[4] In the UK, strains with decreased sensitivity (MIC $\geqslant$ 0·05 mg/l) to ciprofloxacin have been detected since 1989 and treatment failures with ciprofloxacin have been associated with some of these infections.[5,6] More recently reports from the Philippines and Thailand[7,8] have revealed strains with ciprofloxacin MIC of >1 mg/l and in the case of the Philippines at least 10% of strains had a ciprofloxacin MIC of $\geqslant$ 0·25 mg/l. Sentinel studies in the USA have revealed the importation of gonococci with ciprofloxacin MIC of 2 mg/l into Hawaii from SE Asia and also revealed 14% of strains in Ohio to have MICs between 0·13 mg/l and 0·25 mg/l.[9] In 1994 Birley *et al*[10] reported the failure of a five day course of twice daily 250 mg doses of ciprofloxacin in a case of gonorrhoea caught in Spain. This infection was also caused by a strain with high-level resistance to ciprofloxacin (MIC 16 mg/l).

Since 1988 216 strains of gonococci with an MIC of ciprofloxacin $\geqslant$ 0·05 mg/l have been referred to the PHLS Gonococcus Reference Unit and 13 have had an MIC >1 mg/l; none of the strains with reduced sensitivity also had high-level resistance to tetracycline, but 61% were penicillinase producers. Local incidence of ciprofloxacin resistance remains low. In 1994 only 1 of 338 county of Avon isolates was resistant (MIC 1 mg/l); this was not a penicillinase-producer and the infection was contracted in the UK.

This case re-emphasises the importance of culture for cases of gonorrhoea in order to be able to test the antibiotic sensitivities of the organism and thus assist the selection of suitable chemotherapeutic agents. The need for continued vigilance for ciprofloxacin resistance is reinforced, especially where the patient was infected in the Far East.

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Patients' awareness of changes in the Association of British Insurers' guidelines on HIV testing

In July 1994 the Association of British Insurers (ABI) recommended to its members an alteration to the questionnaires for prospective policy holders. Previously, insurers had requested information on whether the applicant had ever taken an HIV/AIDS test. The revised question asked "Have you tested positive for HIV/AIDS".[1] This alteration was precipitated by a campaign co-ordinated by the Terrence Higgins Trust which cited a study commissioned jointly by the ABI and the Department of Health (DOH) which concluded that possibly tens of thousands of people were being put off taking an HIV test because of such questions.[2]

This study was performed to assess the awareness of the alterations to ABI policy and their impact on the attitude to HIV testing in patients attending the John Hunter Clinic. Anonymous questionnaires were given to 130 new patients who were invited to complete them during November–December 1994. To assess the influence of the recommendations, and the possibility of applying for life insurance, on future HIV antibody testing a 5 point